Post- Intercostal Intubation Chylothorax

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Abstract
Chylothorax is a rare condition in childhood. It usually occurs as a post operative complication following cardiac surgery for heart diseases. We report a 16 months old girl with atrial septal defect and severe pulmonary stenosis, hospitalised for empyema and who developed chylothorax following intercostal drainage, an entity which has never been reported.

Key Words
Chylothorax, Medium chain triglycerides, Intercostal drainage

Introduction
Chylothorax generally occurs as a post thoracotomy complication during management of congenital cardiac lesions. However rarely it can occur due to malformation of the pulmonary or thoracic lymphatic systems (1-2). The consequences of chylothorax include fluid and electrolyte disturbances, protein calorie malnutrition, coagulopathy and increased susceptibility to infections (3). An interesting case of chylothorax in a 16 months old child with congenital heart disease as a complication of intercostal drainage is discussed.

Case Report
A 16 months old girl, a known case of otium secundum atrial septal defect and severe valvular pulmonary stenosis presented with high grade fever, poor feeding and breathlessness for one week for which she had received oral antibiotics. Examination revealed a toxic, febrile and malnourished child with evidence of pleural effusion on the left side. The pleural tap revealed a thick purulent exudate (protein content of 6.2 gms% and numerous WBCs/HPF with 75% polymorphs and staining revealed Gram positive cocci) She was managed with parenteral antibiotics and intercostal drainage. The intercostal tube drained 500 ml of frank yellow pus on day one which gradually reduced to 50 ml on third day. She became afebrile after three days but on the fourth day, the drainage fluid was observed to be thin, copious, milky white and its volume increased to 700-800 ml/day. A clinical diagnosis of chylothorax was now made and confirmed by reanalysing the fluid: proteins 4.5 gms%, triglycerides 1255 gms% and lymphocytic predominance. CT chest showed normal vascular structures, lung parenchyma and did not reveal any mediastinal or hilar lymphadenopathy. Lymphangiography could not be done as the facilities for same were not available. Child was managed with medium chain triglyceride enriched milk. The amount of fluid drained decreased after seven days, intercostal tube was taken out on 15th day of admission and she was discharged after 35 days of hospitalisation. A follow up after six weeks revealed status quo.

Discussion
Chylothorax is the accumulation of lymph fluid or chyle in the pleural space. It may be traumatic or non traumatic in origin. Nearly half of the cases of traumatic chylothorax are postoperative following cardiac surgery in congenital heart diseases (4). The etiopathogenesis of such chylothorax is considered to be due to direct injury to the main thoracic duct/viable lymphatic pathways/accessory lymph channels during operative approaches or
disruption of microscopic lymphatic channels with lymphatic hypertension (5). The other causes of trauma include direct laceration of thoracic duct from penetrating injury or fracture. The common non-traumatic causes include lymphadenopathy and intrathoracic tumours. Less common causes are lymphangiomatosis, restrictive pulmonary diseases, thrombosis of duct or subclavian vein and congenital anomalies of thoracic duct (6). There is generally a latent period of 2-10 days between trauma to the thoracic duct or its tributaries and the development of chylous effusion. However in rare instances it may be prolonged to even a few weeks or months. The reasons attributed to the delayed occurrence in these patients are decreased oral intake and limited patient movement during the early post operative period which leads to a slow accumulation of chyle (7). The reported case had developed chylothorax, clinically attributable to thoracic duct injury, 4 days after intercostal drainage.

The management of a chylothorax is mainly conservative. It includes chest tube drainage, use of medium chain triglycerides, low fat enteral diets, parenteral nutrition and somatostatin. Oral medium chain triglyceride rich diet and adequate drainage of pleural space should comprise the early management in these cases. Such a diet not only provides adequate nutrition but also decreases the lymph leak which supports healing of lymphatics. If effusion does not decrease, oral or enteral feeding should be stopped and total parenteral nutrition should be initiated (4,5). In a series of 15 patients of chylothorax, Narumon et al reported that twelve patients responded to a medium chain triglyceride rich diet, two cases resolved after switching to total parenteral nutrition and only one case needed surgery (7). Recently, subcutaneous or intravenous infusion of octreotide has been used as a safe treatment that helps avoiding surgical intervention. Somatostatin may act directly on vascular receptors to reduce lymph excretion. Since lymph flow in the thoracic duct mainly depends on the state of splanchnic circulation and intestinal motility, it may have a role in reducing lymph flow (8).

Surgical management includes pleurodesis or ligation of the main duct adjacent to leaking lymphatic lymphatics. The timing of surgery is not defined as uniformly as is the initial therapy. Most authors recommend that surgical treatment should be performed if there is no response to conservative treatment for 4 weeks. Early surgery however shortens the hospitalisation time and reduces the nutritional and infectious complications (1). Our patient was managed conservatively with intercostal drainage and medium chain triglyceride rich diet and recovered fully.

**Conclusion**

Chylothorax following intercostal drainage has not been reported so far to the best of our knowledge. The present case initially presented as pyothorax which showed an early response to antibiotics and intercostal drainage. On fourth day there was a visible change in appearance and volume of fluid which was now thinner and milky. The change from pyothorax to chylothorax could be attributed in this instance to trauma to the thoracic duct as a result of intercostal tube insertion. Normally the thoracic duct ascends through the aortic opening in the diaphragm on the right side of the descending aorta. It gradually crosses the median plane behind the esophagus and reaches the left border of the 4th thoracic vertebra. It is therefore unlikely to be traumatised by intercostal tube on the left side unless it is pushed very high above in the pleural cavity. The other possibility could be trauma to the malformed or aberrantly placed lymphatic duct. The presence of an underlying congenital heart defect in this patient could well be associated with another malformation.

**References**